

**UNITED STATES DISTRICT COURT  
DISTRICT OF NEW JERSEY  
CAMDEN VICINAGE**

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**IN RE  
PAULSBORO DERAILMENT CASES**

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**MASTER DOCKET NO.:  
1:13-CV-784 (RBK/KMW)**

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**RONALD MORRIS AND KRISTEN  
PICKEL**

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**Plaintiffs,**

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**CIV NO. 1:13-03244 (RBK/KMW)**

:

**v.**

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:

**CONSOLIDATED RAIL  
CORPORATION, *et al.*,**

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**Defendants.**

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**PLAINTIFF'S BRIEF IN OPPOSITION TO DEFENDANTS'  
MOTION TO EXCLUDE THE EXPERT REPORT AND  
TESTIMONY OF OMOWUNMI OSINUBI, M.D. [ECF #726]**

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## **INTRODUCTION**

On November 30, 2012, a train derailed while crossing the Paulsboro Moveable Bridge as a result of the tortious conduct of Defendants Consolidated Rail Corporation, Norfolk Southern Railway Company, and CSX Transportation, Inc. (collectively, “Defendants”). One of the tank cars breached, releasing well over 20,000 gallons of vinyl chloride gas into the air. The gas spread throughout the town.

Plaintiff Ronald Morris (“Plaintiff” or “Morris”) was one of the many who were exposed, and he suffered immediate respiratory distress, requiring multiple trips to the hospital on the day of the derailment. His medical problems did not end there: Morris continues to experience breathing difficulties and shortness of breath, throat irritation, sleep problems, and emotional distress.

Morris retained Omowunmi Osinubi, M.D. as a causation expert in his suit against Defendants, and for purposes of recommending whether medical monitoring would be appropriate. Defendants have moved to exclude all testimony of Dr. Osinubi based on a litany of purported defects in her methodology, all of which go to the weight, rather than the admissibility of Dr. Osinubi’s testimony.

Because Dr. Osinubi’s testimony satisfies the standards of *Daubert* and Fed. R. Evid. 702, Defendants’ motion to exclude must be denied.

## **BACKGROUND**

### **I. The Derailment Resulted in a Massive Exposure to Vinyl Chloride.**

Raelynn Stevenson was drinking her morning cup of coffee at her home when she witnessed the derailment. She described a vapor cloud that appeared right after the train cars derailed that looked like “the dust that came up the street that you saw on TV on 9/11. That’s what it looked like coming up to my house. It looked like the 9/11 dust.” *See* Declaration of David Cedar in Support of Plaintiffs’ Opposition to all *Daubert* Motions and all Motions to Dismiss Personal Injury, Medical Monitoring and Emotional Distress Claims (“Cedar Dec.”) Exhibit A (NTSB Interview of Raelynn Stevenson (“R. Stevenson Statement”)), at p. 9:15-6; 10:6-16. Before the accident, the weather “was crystal clear. It was a beautiful morning.” *Id.*, p. 11:17-8

Conrail’s engineer confirmed that “as soon as the top of the bridge went down it was almost instantaneously that the fog bank came up out of the Mantua Creek.” *See* Cedar Dec. Exhibit D (Deposition Transcript of Mark Mather (“Mather Dep.”)), p. 103:12-14. Conrail’s engineer described the size of the vapor cloud: “Pretty much the whole neighborhood . . . had a fog.” *Id.* at 113:19-21.

The official Paulsboro police report stated: “as [Patrolman Rodney Richards] was speaking with [Conrail’s] conductor, [he] noticed a smoky fog start to swarm the immediate area and become very thick. The smoky substance that quickly

surrounded [him] caused a reaction that made [him] cough several times.” Cedar Dec. *See* Exhibit F (Paulsboro Police Report), p. 2.

The recorded communications of the first responders further supports the immediate presence of toxic fog:

7:01 “It’s [the train] is spewing out all kinds of gas.” (Gloucester County 911 call)

7:05 “Rail cars or tank cars have been pierced and have leaked out all of their contents into the creek. The creek is full of vapors from these cars.” (Channel 3 fire Ops.)

7:06 “It’s a major emergency, bridge collapsed and major hazards, potentially life-threatening...I have an odor out here that they are not familiar with. This odor is hazardous. Hazard released.” (Zone 3 Police Radio Channel)

*See* Cedar Dec. Exhibit G (Timeline of Events and Communications on November 30, 2012).

The ruptured tank car contained about 25,000 gallons, or about 177,000 pounds, of vinyl chloride. The National Oceanic and Atmospheric Administration (NOAA) ran an Area Locations of Hazardous Atmospheres (“ALOHA”) model of what the exposures would be if the entire amount were released through a hole 12 inches in diameter over a time period of two minutes. This model showed that the “toxic threat zone” would extend out two miles from the release area. *See* Cedar Dec. Exhibit J (NOAA ALOHA Model for Paulsboro, NJ). The toxic threat zones are assessed in terms of Acute Exposure Guideline Levels (“AEGLs”) established by the National Advisory Committee managed by the Environmental Protection

Agency.

Severity Tier	Vinyl Chloride Air Concentration (ppm)			Definition
	Exposure for 10 minutes	Exposure for 30 minutes	Exposure for 60 minutes	
AEGL-1	450	310	250	The airborne concentration of a substance above which it is predicted that the general population, including susceptible individuals... ...could experience notable discomfort, irritation, or certain asymptomatic nonsensory effects. However, the effects are not disabling and are transient and reversible upon cessation of exposure.
AEGL-2	2,800	1,600	1,200	...could experience irreversible or other serious, long-lasting adverse health effects or an impaired ability to escape.
AEGL-3	12,000	6,800	4,800	...could experience life-threatening health effects or death.

*See Cedar Dec. Exhibit I (NJ DOH Air Quality Consultation).*

The highest threat zone, corresponding to AEGL-3 at 4,800 ppm, would extend 1,383 yards, or about 0.8 mile, from the spill site; the next highest threat zone, corresponding to AEGL-2 at 1,200 ppm, would extend from 0.8 to 1.2 miles away from the spill site; and the last threat zone, corresponding to AEGL-1 at 250 ppm, would extend from 1.2 to 2.0 miles away from the spill site. *See Cedar Dec. Exhibit P . An Interagency Modeling and Atmospheric Assessment Center (“IMAAC”)* model similarly estimated that the AEGL-3 zone would be about 0.5 mile in diameter and the total toxic threat zone about 1.0 mile in diameter. *See Cedar Dec. Exhibit K (IMAAC Model).*

Conrail’s contractor determined that when turned to gas, the entire 80,000 kg

of vinyl chloride would spread over a surface area of 30,000 square meters and one meter thick. *See* Cedar Dec. Exhibit R (ARCADIS US's Vinyl Chloride Model). This means that an area encompassing approximately three square blocks of Paulsboro would be 100% vinyl chloride, or, in the alternative, that there would be enough vinyl chloride to contaminate an area of 300 city blocks to a level of 10,000 parts per million if equally disbursed.

## **II. Paulsboro Refinery Vinyl Chloride Testing Results**

Paulsboro Refinery personnel arrived at the scene of the derailment at about 8:30 a.m. with Photo Ionization Detectors ("PID"). They found that the levels of vinyl chloride in the air were so high that they were unable to "zero" out their equipment. Without being able to "zero" out the PIDs first, the readings on these devices yielded results of 631, 694 and 760 ppm. As analytic chemical expert, Dr. Brian Buckley, explains, these readings need to be multiplied by 1.9 to obtain a vinyl chloride equivalent. *See* Cedar Dec. Exhibit M (Report of Brian Buckley ("Buckley Report")), p. 1.

About ten minutes later, the Paulsboro Refinery employees walked a few blocks away to Delaware Street and Billings Avenue to try to zero out their instruments, but instead obtained readings well in excess of 100 ppm. *Id.* at 2. The same meters later on had negative readings ranging from -40 to -60 ppm which is, of course, impossible. *Id.* Dr. Brian Buckley reviewed the Paulsboro Refinery

data and opined that the actual vinyl chloride levels present in the air at the time the Paulsboro Refinery readings were taken were “greatly in excess of the readings recorded by the device.” *Id.* at 3. Due to being unable to properly zero the device out and to the excessive saturation of the chemical, the readings from the Paulsboro Refinery PIDs could reasonably be expected to result in much lower levels than was actually present. *Id.*

### **III. New Jersey Department of Health Consultations**

The New Jersey Department of Health (“DOH”) performed a “Health Consultation” on air quality in Paulsboro following the derailment which concluded that “[b]ased on modeled estimates and monitoring, peak air concentrations . . . exceeded the EPA’s Acute Exposure Guidance Levels (AEGL) for one hour exposure that are associated with reversible health effects (AEGL-1: 250 ppm) and possibly disabling effects (AEGL-2: 1,200 ppm) or life threatening effects (AEGL-3: 4,800 ppm).” Cedar Dec. Ex. I at 11.

### **IV. Odor Detection and Threshold**

Many in Paulsboro complained of the sickly sweet odor of vinyl chloride on the day of the chemical spill. One of the complainants, Stephanie Esposito, a Fox 29 news reporter, tweeted at 9:13 a.m. on November 30th that she “just took a walk down to the roadblock and definitely smell[ed] something sweet.” She described the odor as “a pungent smell [that] hits you like a brick wall when you

walk into it". She was able to smell this odor even though she had a cold. *See* Cedar Dec. Exhibit Y. Ptl. Richards, who had worked in Paulsboro since 2006, testified that the odor of chemical was distinct from the typical smells in the town. He too described it as a "pungent smell". *See* Cedar Dec. Exhibit S, p. 31-32.

Dr. Maria Kent, who is a family practitioner in Paulsboro, testified that multiple patients were concerned about their vinyl chloride exposure and reported complaints of nausea, headaches, wheezing, confusion, dizziness and dry throat. *See* Cedar Dec. Exhibit T pp. 41-43. A number of these patients also complained of smelling some kind of chemical odor that day. *Id.*

The Agency for Toxic Substances and Disease Registry ("ATSDR") also reports the odor threshold for vinyl chloride to be "about 3,000 ppm" noting that it varies significantly among individuals. *See* Cedar Dec. Exhibit V.<sup>1</sup> An odor threshold test performed by Union Carbide on a panel of experts with an average of ten years experience in odor detection and evaluation found that the odor threshold for vinyl chloride was commonly 2,000 ppm, although two of the most sensitive panelists could detect a faint odor at 1,200 ppm. *See* Cedar Dec. Exhibit W.

The New Jersey Department of Health took two surveys of residents of Paulsboro following the train wreck. The first was an in person "door to door"

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<sup>1</sup> The NJDOH Hazardous Substance Fact Sheet states "Odor threshold = > 3,000 ppm."

survey and the second was a mailed survey. 50% of the adults in the in-person survey reported smelling or tasting unusual odors; in the mailed survey 69% of households reported that at least one member of the household smelled or tasted an unusual odor. *See* Exhibit X (NJ DOH Health Consultation), p. 8. Among those who reported smelling or tasting an odor in both the in-person and mailed surveys, there were higher frequencies of reported new or worsening symptoms. Also in both surveys, the most frequently reported symptoms among those who smelled an odor were headache, coughing, irritation of the nose and throat, dizziness, irritation or pain or burning of eyes, and difficulty breathing. *Id.* The DOH concluded that:

The symptoms commonly reported are consistent with what is known to occur from exposure to vinyl chloride, specifically headache, irritation of the eyes, nose, throat and lungs, coughing, nausea, and dizziness or lightheadedness. ... [T]here was a similar pattern of reported symptom frequencies between the in person and mailed surveys, as well as with the findings of surveys of emergency responders.

*Id.* at 12.

The report also found that “symptoms were most commonly reported from evacuated areas and areas within one block of evacuated areas and were least frequent in areas farther than 3,500 feet from the derailment location.” *Id.*

## **V. Vinyl Chloride Health Information**

The New Jersey DOH has a hazardous substance fact sheet for vinyl chloride, which states:

Vinyl chloride is a CARCINOGEN in humans. There may be no safe level of exposure to a carcinogen.

The acute health effects include the following:

- Exposure to **Vinyl Chloride** can severely irritate and burn the skin and eyes with possible eye damage. Contact with the *liquid or gas* can cause frostbite.
- Inhaling **Vinyl Chloride** can irritate the nose, throat and lungs causing coughing, wheezing and/or shortness of breath.
- **Vinyl Chloride** can cause headache, nausea, vomiting, dizziness, fatigues, weakness and confusion. Higher levels can cause lightheadedness and passing out.

Cedar Dec. Exhibit Y. OSHA's permissible exposure limit for vinyl chloride is 1 ppm averaged over an 8 hour day. The short-term exposure limit is 5 ppm, not to be exceeded during any 15-minute period. *Id.* OSHA recommends medical monitoring consisting of liver function tests, chest X-rays and lung function tests for workers exposed to 0.5 ppm of vinyl chloride. Cedar Dec. Exhibit Z.

The International Agency for Research on Cancer's (IARC) Monograph on the Evaluation of Carcinogenic Risk to Humans found sufficient evidence that vinyl chloride causes angiosarcomas of the liver and hepatocellular carcinomas (HCC). *See* Cedar Dec. Exhibit AH, p. 425. The same report cited numerous studies which "found evidence of a significant association between exposure to vinyl chloride monomer (VCM) and mortality from liver cirrhosis. *Id.* at 327 (citing The European Multicentric Study); *id.* at 328 (citing Pirastu, et al., 2003). Mastrangelo in 2004 also reported "an association between exposure of VCM and

both liver cirrhosis and [hepatocellular carcinoma].” *Id.* at 329; *see also id.* at 349 (a Cross Sectional Study of Hepatocellular Carcinoma in Italy found an association between exposure to VCM and both liver cirrhosis and HCC in vinyl chloride workers). IARC concluded “together with the observation that vinyl chloride increases the risk for liver cirrhosis, which is a known risk factor for hepatocellular carcinoma, these findings provide convincing evidence that vinyl chloride causes hepatocellular carcinoma as well as angiosarcoma of the liver.” *Id.* at 422.

In addition, the Material Safety Data Sheet (MSDS) of Oxyvinyl issued for this very shipment of vinyl chloride risks among potential health effects states the following:

Inhalation: May cause respiratory tract irritation. Several minutes of exposure to high, but attainable concentrations (over 1,000 ppm) may cause difficulty breathing, central nervous system depression and symptoms such as: ataxia or dizziness, drowsiness or fatigue, loss of consciousness, headache, euphoria and irritability, visual or hearing disturbances, nausea, memory loss. Prolonged high concentration exposure may cause unconsciousness or death. Cardia: Acute intoxication may cause irregular heartbeats.

*See Cedar Dec. Exhibit AB, MSDS on Vinyl Chloride (Monomer), p. 10.*

Mastrangelo, et al.’s 2004 study explicitly concluded that “VCM exposure appears to be an independent risk factor for [hepatocellular carcinoma] and [liver cirrhosis].” *See Cedar Dec. Exhibit AJ, Mastrangelo, et al., Increased Risk of Hepatocellular Carcinoma and Liver Cirrhosis in Vinyl Chloride Workers: Synergistic Effect of Occupational Exposure with Alcohol Intake, Vol. 112 (2004),*

p. 1192.

# **VI. Statement of Facts Specific to Plaintiff Ronald Morris.**

On the morning of November 30, 2012, Morris headed to work at a construction site. He was passing through Paulsboro at North Commerce Street when, at 7:45 a.m., he encountered a thick cloud. The fog was so dense that he could not see his hand in front of his face and could only drive at 2 miles per hour. After passing through the fog he then encountered another cloud on Crowne Point Road. His skin and eyes started to burn, but he continued to drive slowly through the cloud to get to his job site. He estimated it took him about 15 minutes to get through the cloud, due to the poor visibility.<sup>2</sup>

After arriving at work he developed trouble breathing, skin and eye irritation, burning and an upset stomach. He was taken to the emergency room at Underwood Hospital where he was stripped of his clothing and placed into decontamination. *See* Cuker Dec. Ex. A, Osinubi Report March 5, 2015 at p. 4.

He reported chest pain rated at 8/10, an unsteady gait and vomited on his arrival at the hospital. After leaving Underwood, Morris went to the American Legion to meet his father. He was so shaken by the experience that he had five to six beers at the American Legion. After going home he developed severe cough spells and coughed up blood. He was taken by ambulance and given oxygen

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<sup>2</sup> *See* March 5, 2015 Report of Dr. Osinubi for Ron Morris, p.4. Cuker Dec. Ex. A; ER record of Underwood Hospital. Cuker Dec. Ex. B

treatment on his way to Underwood a second time that day. The ambulance personnel reported he was very anxious because of his difficulty breathing; however, he left the emergency room without being seen by the doctor because he felt the paramedical staff did not take his condition seriously. *See* Cuker Dec. Ex. C, Gloucester County EMS report dated November 30, 2012.

Morris continued to experience shortness of breath, cough, headaches, nausea, and anxiety and was seen by Dr. Vernon on December 6, 2012, who ordered a urine test. The urine test drawn on December 6 was positive for vinyl chloride metabolite, which was remarkable since the vinyl chloride would not have been expected to remain in his body more than 24 hours after the exposure. *See* Cuker Dec. Ex. D, Report of Michael I. Greenberg dated April 16, 2015, at p. 18. This finding was plainly abnormal. *See* Cuker Dec. Ex. E, Dr. Osinubi Dep. at 217:4-20. A Spirometry report performed by Dr. Vernon was abnormal with signs of moderate obstruction, an indicator that his “lung age is 108 years.” *See* Cuker Dec. Ex. A, at p. 10. His FEV1 was 48% of predicted, indicating acute discomfort that would not be expected in a 20-pack year smoker.

*See* Cuker Dec. Ex. E, Osinubi dep. at 209:6-210:10. Dr. Vernon heard decreased breath sounds throughout all lung fields and diagnosed reactive airway disease/bronchospasm and prescribed continued nebulizer treatment. The last time Morris saw Dr. Vernon on January 14, 2013, he still had intermittent dizziness,

coughing and difficulty breathing. *See* Cuker Dec. Ex. A, Dr. Osinubi March 5, 2015 report, p. 11.

Dr. Osinubi conducted a 90-minute telephone interview of Mr. Morris on January 23, 2015, and a physical examination on February 20, 2015. On examination she heard crepitations in the mid/lower chest bilaterally which did not clear with cough. She ordered a high resolution CT scan which found focal areas of ground glass opacities. A cardio respiratory exercise study was abnormal. Morris's exercise was limited by reduced maximal oxygen consumption at 71% of his maximum predicted level. Dr. Osinubi diagnosed chronic dyspnea on exertion resulting in physical activity limitations from intense and substantial exposures to vinyl chloride. She stated that this "diagnostic finding corroborates Mr. Morris's reports that since his vinyl chloride exposure he is no longer able to the typical physically demanding activities required for his job as a construction worker." *Id.* at p. 29. She testified that these abnormal findings are seen "in people who were exposed to high levels of irritants and then present with shortness of breath." *See* Cuker Dec. Ex. E, Dep of Dr. Osinubi May 8, 2015 dep. at 227:9-24.

Mr. Morris's CT scan demonstrated fatty liver disease (steatohepatitis) which could result from both alcohol and vinyl chloride exposure.

Dr. Osinubi stated:

Mr. Morris had substantial exposures to vinyl chloride and then used substantial alcohol on the same date following which he

ended up in the emergency room with a recurrence of his vinyl chloride toxicity symptoms and respiratory distress. The liver enzymes that metabolize alcohol are used for metabolizing solvents and xenobiotics such as vinyl chloride. This may explain in part why he had significant levels of vinyl chloride in his urine six days after exposure, as he likely had significant body burden of vinyl chloride. This likely predisposes Mr. Morris to a higher likelihood of end organ damage and his long term health effects from vinyl chloride exposure.

Cuker Dec. Exhibit “A” at p. 30.

Dr. Osinubi concluded that Morris “has a persistent chronic cough, shortness of breath on exertion, fatigue and body aches and joint pain, all of which adversely impact his activities of daily living,” and that “[o]bjective diagnostic assessments confirm abnormal ground glass findings on his chest CT scan; steatohepatitis which is likely related to his dual exposures to vinyl chloride and alcohol; abnormal limitation to his exercise capacity and finding suggestion of mitochondrial myopathy.” *Id.* at 32.

Dr. Osinubi wrote a second addendum report on May 7, 2015, which incorporated the pollutant dispersion model of Dr. Panos Georgopoulos. Dr. Osinubi reported that Dr. Georgopoulos’s dispersion modeling showed Morris was in “the AEGL-3 toxic zones, with exposures potentially as high as 4,000 ppm or more.” *See* Cuker Dec. Ex. F at 17. The modeling also estimated vinyl chloride decay product concentrations from 100 to 1,000 mg/m<sup>3</sup>, which would more likely than not include 50% hydrochloric acid (HCL). This would substantially exceed

the AEGL-2 level for disabling symptoms for hydrochloric acid and possibly exceed the AEGL-3 level<sup>3</sup>. Dr. Osinubi noted that both hydrochloric acid and formaldehyde, the primary decay products of vinyl chloride, irritate the respiratory tract, cause coughing, shortness of breath, and irritate the skin. *See* Cuker Dec. Ex. F, at 6-7. She concluded that “the acute symptoms experienced by Paulsboro residents, inclusive of Ms. Alice Breeman and Mr. Ronald Morris, in the aftermath of the massive vinyl chloride release is consistent with what is known as health effects of vinyl chloride and its decay products, inclusive of hydrochloric acid, formaldehyde and other(s).” *Id.* at p. 12.

Dr. Osinubi summarized her opinion on Mr. Morris as follows:

To the extent that his history states his dyspnea started, at least significant dyspnea started after the exposure to vinyl chloride and he had objective evidence of airway-airway obstruction shortly after the exposure, he was acutely sick from the exposure and as of December 6 he had evidence of airway obstruction, I would say the exposure to vinyl chloride is contributory to a material degree to a shortness of breath on exertion, in addition to the smoking history.

*See* Cuker Dec. Ex. E, Osinubi Dep. at 229:18-230:8.

### **ARGUMENT**

“The Rules of Evidence embody a strong and undeniable preference for admitting any evidence which has the potential for assisting the trier of fact.”

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<sup>3</sup> The 10-minute AEGL-2 level is 100 mg/m<sup>3</sup> for HCL and 2800 ppm for vinyl chloride.

*Kannankeril v. Terminix Int'l Inc.*, 128 F.3d 802, 806 (3d Cir. 1997) (citing *Holbrook v. Lykes Bros. S.S. Co.*, 80 F.3d 777, 780 (3d Cir. 1996)); *see also* Fed. R. Evid. 402 (“Relevant evidence is admissible.”). If expert evidence is admissible, the trier of fact will determine the proper weight to give it. *Maloney v. Microsoft Corp.*, 2011 U.S. Dist. LEXIS 127870, at \*6-7 (D.N.J. Nov. 4, 2011).

In considering pre-trial challenges to expert testimony, Rule 702 has “three major requirements: (1) the proffered witness must be an expert, *i.e.*, must be qualified; (2) the expert must testify about matters requiring scientific, technical or specialized knowledge; and (3) the expert’s testimony must assist the trier of fact.” *Pineda v. Ford Motor Co.*, 520 F.3d 237, 244 (3d Cir. 2008).<sup>4</sup> “A district court’s inquiry under Rule 702 is ‘a flexible one’ and must be guided by the facts of the case.” *ZF Meritor, LLC v. Eaton Corp.*, 696 F.3d 254, 294 (3d Cir. 2012).

#### **I. Dr. Osinubi did not rely on the NTSB’s report.**

Defendants falsely claim that Dr. Osinubi “*relied* upon the NTSB Accident Report and Factual Report as the underlying basis for *all* of her opinions in this case.” Defs. Br. at 6 (emphasis added). Although Dr. Osinubi reviewed the NTSB’s Accident Report, she did not “rely” on it to support her substantive opinions. On the contrary, she relied on a mountain of other exposure-levels evidence (cited above), including the Air Quality Consultation of the New Jersey

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<sup>4</sup> Defendants do not challenge Dr. Osinubi’s qualifications.

Department of Health and the Air Modeling Report of Dr. Georgopoulos, both of which confirm that the levels were, in fact, very high. *See, e.g.*, Cuker Dec. Exhibit “A”.

During her deposition, Dr. Osinubi referenced the NTSB, the New Jersey DOH, the EPA, ALOHA modeling, and Dr. Georgopoulos collectively merely to note the universal agreement of credible sources that exposure levels were very high. *See* Cuker Dec. Ex. G, Dr. Osinubi Dep. at 91-92. Her casual reference to the NTSB is inconsequential to the opinions she will offer at trial. Defendants’ argument for exclusion under 49 U.S.C. § 1154(b) is thus a red herring and should be summarily rejected.

**II. Dr. Osinubi used well-established and generally accepted methodology to opine on general causation.**

Dr. Osinubi’s general causation methodology is not only logical but generally accepted in her field and the courts. She looked to see if there was a high exposure, and (since there was such an exposure) whether the symptoms experienced by the plaintiff began shortly after the exposure and were consistent with what would be expected to result from it. *See* Osinubi Dec. at ¶ 3. This was the identical methodology used by the New Jersey Department of Health in its own health consultation for Paulsboro and accepted by Dr. Greenberg (Defendants’ expert) himself. *See Id.* at ¶ 5.

**A. Dr. Osinubi’s general causation opinion easily survives**

under *Kannankeril v. Terminix Int'l*, 128 F.3d 802 (3d Cir. 1997).

In *Kannankeril*, the plaintiff claimed she had a cognitive impairment caused by exposure to pesticides applied by Terminix. The trial court struck the expert's causation testimony on grounds that there was no air-testing sufficient to support the expert's opinion about the plaintiff's exposure and that the expert's opinion on causation was unreliable and unsupported by fact, the same arguments made by Defendants here. The Third Circuit reversed. In discussing the exposure, the Third Circuit rejected the notion that the plaintiff's expert had to rely on ambient air tests (which were not conducted until 9 months after the application of pesticides), and found it sufficient for the expert to look at Terminix's application records showing when, how much and where pesticide had been applied.

*Kannankeril*, 128 F.3d at 808-809. Critical to the instant motion, the Third Circuit in *Kannankeril* held that "***all factual evidence*** of the presence of the chemicals in the residence should be relevant in forming an expert opinion of causation." *Id.* at 809 (emphasis added). The Third Circuit's holding cements the principle that *Daubert* reliability determinations must be made upon consideration of the full evidentiary record, which will dictate whether or not certain exclusionary principles are apt.

In this case, although there are no precise measurements of Morris's exposure, it is undisputed that 23,000 gallons of vinyl chloride were released into

the environment and that this amount of vinyl chloride would fill up a cloud over 27,000 cubic meters in size of 100% Vinyl Chloride. This is more than analogous to the “application records of how much pesticide was applied” in the *Kannankeril* case. If that were not enough, there is substantial evidence that Morris drove through a vinyl chloride cloud, immediately experienced symptoms and a strange taste, and had an abnormal urine test *positive for a vinyl chloride metabolite* six days later. And then there is Dr. Georgopoulos’s model and the NJ DOH report, both of which document extensive levels of exposure and demonstrate that Morris’s geographic position on November 30, 2012 subjected him to those high levels.<sup>5</sup>

In *Kannankeril*, the expert’s methodology was indistinguishable from that employed by Dr. Osinubi here: “The temporal relationship and nature of her complaints led me to conclude that with reasonable medical certainty the cause of Dr. Kannankeril’s central nervous system manifestations of toxicity is exposure to Dursban.” *Kannankeril, supra*, 128 F.3d at 805. The Third Circuit concluded that because the plaintiff’s expert had based his opinion on the plaintiff’s medical

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<sup>5</sup> Because this extensive exposure evidence supports Dr. Osinubi’s opinions, there is no need to discuss Defendants’ pointless attack on the ALOHA model. *See* Defs. Br. at 12-15. In addition, we respond to the attack on Dr. Georgopoulos’s air model in our opposition to Defendants’ motion (ECF No. 737) to exclude his testimony. It suffices to say here that “when direct measurements cannot be made, exposure can be measured by mathematical modeling, in which one uses a variety of physical factors to estimate the transport of the pollutant from the source to the receptor.” Reference Manual on Scientific Evidence, p. 424 (2d ed. 2000).

records and reports of the volume of pesticide applied and his general experience, general medical knowledge, standard text books, and standard references, the experts “opinion on causation has a factual basis and supporting scientific theory.”

There is a legion of case-holdings in line with Dr. Osinubi’s methodology – that an acute exposure closely followed by symptoms known to result from that exposure provides good grounds for an expert’s opinion on causation. *See, e.g., Thomas v. CMI Terex Corp.*, 2009 U.S. Dist. LEXIS 86623, at \*40 (D.N.J. Sept. 21, 2009) (Simandle, J.) (“The question of causation can be resolved by a doctor without even medical testing, where the temporal proximity between an accident and the subsequent injury make the accident the most probable cause of the injury.”) (and collecting cases). In *Winnicki v. Bennigan’s, et al.*, 2006 U.S. Dist. LEXIS 5568 (D.N.J. Feb. 9, 2006) (Greenaway, Jr.), for example, the court evaluated expert testimony in a case in which the plaintiff ate a Caesar salad the night before she became sick with acute gastrointestinal dysfunction, which led to kidney failure and death. Although the expert could never determine exactly what was wrong with the salad, he opined that the salad was the cause of the condition using a differential diagnosis and temporal relationship. The court denied the defendant’s motion to exclude the expert, citing Third Circuit precedent (*e.g., Kannankeril*) accepting “medical testimony that relies heavily on a temporal relationship between and illness and a causal event.” *Id.* at \*46.

**B. Causation can be established in the absence of a precise measurement of Morris's level of exposure, particularly where, as here, there is abundant evidence of substantial exposure.**

The only distinction between the defense expert's (Dr. Greenberg) methodology and that of Dr. Osinubi is that Dr. Greenberg would require idiosyncratic measurement of exposure to conclude that there is a completed exposure pathway. Osinubi Dec. at ¶¶ 4, 6. But that was not required by the New Jersey DOH, nor is it required by the pertinent case law.<sup>6</sup> As the Third Circuit explained in *Heller v. Shaw Indus., Inc.*, 167 F.3d 146, 157 (3d Cir. 1999), "even absent hard evidence of the level of exposure to the chemical in question, a medical expert could offer an opinion that the chemical caused plaintiff's illness." As another court has explained, in rejecting a railroad's *Daubert* challenge:

We disagree with CSX that in order to validate the testimony of the medical experts, Moody was required to prove the precise dosage of solvents to which he was exposed and the precise level required to have a harmful effect on human beings. \* \* \* We believe that Moody presented sufficient evidence both as to his level of exposure and that necessary to cause his toxic encephalopathy. He presented testimony concerning how often he used the offending solvents and the duration of his exposure. He further explained the physical symptoms that he suffered while working with the solvents. While not quantitatively specific, the expert testimony supports the conclusion that Moody's exposure, under the circumstances described, and his

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<sup>6</sup> Of course, record evidence already shows that, according to the New Jersey DOH, "[i]n Paulsboro there *was* a completed exposure pathway to Vinyl Chloride in the hours and days following the derailment . . ." Osinubi Dec. at ¶ 7 (quoting NJ DOH Air Quality Health Consultation, p. 6) (emphasis modified).

length of the exposure, are sufficient to cause his toxic encephalopathy.

*CSX Transp., Inc. v. Moody*, 2007 Ky. App. LEXIS 208, at \*18-19 (Ky. Ct. App. July 13, 2007); *see also Whitlock v. Pepsi Ams.*, 527 Fed. App'x 660, 661-662 (9th Cir. 2013) (“Plaintiffs’ **probable** ingestion of TCE-contaminated groundwater,” coupled with the fact “that the alleged TCE and chromium exposure levels were ‘within [a] reasonable range of that known [from several studies] to induce’ the alleged injuries” was sufficient for expert testimony to satisfy *Daubert*; “Whether [that testimony] proves causation is not a question of admissibility.”) (emphasis added); *Louderback v. Orkin Exterminating Company*, 26 F. Supp. 2d 1298, 1306-07 (D. Kan. Oct. 14, 1998) (as long as expert considered facts of plaintiff’s exposure, the temporal relationship between exposure and disease, the plaintiff’s medical records and history of disease, then an expert’s opinion on causation is considered reliable and clearly “has a factual basis and supporting scientific theory” even when there is no specific evidence of exposures in excess of the ACGIH threshold level or the EPA reference dose); *Harris v. Peridot*, 313 N.J. Super 257, 298 (N.J. Super. Ct. - App. Div. 1998) (holding that an expert could reasonably consider the fact that the injuries sustained are consistent with a high level of exposure on “both sides of the equation”, i.e. as additional evidence supporting the conclusion that the exposure was substantial).

**C. The Bradford Hill factors are not a *per se* requirement, and**

**do not apply in cases focusing on acute exposure.**

While Defendants focus almost exclusively on the Bradford Hill methodology, it does not govern a case of acute exposure causing acute injury. *See* Osinubi Dec. at ¶ 1. As noted, *supra*, courts recognize that a strong temporal relationship and immediate symptomology can support a conclusion of causation. *See, e.g., In re Stand 'N Seal Prods. Liab. Litig.*, 623 F. Supp. 2d. 1355, 1371-72 (N.D. Ga 2009) (causation opinion that exposure caused chemical pneumonitis survived *Daubert* challenge because strong temporal relationship between exposure and acute onset of respiratory symptoms, despite lack of dose/response data); *In re Ephedra Prods. Liab. Litig.*, 2007 U.S. Dist. LEXIS 74914, at \*7 (S.D.N.Y. Oct. 5, 2007), *vacated and remanded on other grounds by Giordano v. Market Am., Inc.*, 289 Fed. App'x 467, 469 (2d Cir. 2008) (“The close temporal proximity between Ms. Stafford’s stroke and her use of ephedra, coupled with the general-causation evidence about ephedra’s rapidly acting biological effects (in contrast to asbestos), permit a jury to infer that the dose she ingested was sufficient to be considered a substantial factor in causing her stroke.”); *see also Cavallo v. Star Enter.*, 892 F. Supp. 756, 774 (E.D. Va. 1995) (“[T]here may be instances where the temporal connection between exposure to a given chemical and subsequent injury is so compelling as to dispense with the need for reliance on standard methods of toxicology.”); *accord Nat’l Bank of Commerce*

*v. Dow Chemical Co.*, 965 F. Supp. 1490, 1525 (E.D. Ark. 1996).

Sir Bradford Hill himself recognized that his viewpoints could be irrelevant in the case of acute exposure to indisputably toxic chemicals. *See* Osinubi Dec. at ¶ 1 (“A particular and perhaps extreme physical environment cannot fail to be harmful. A particular chemical is known to be toxic to man therefore suspect on the factory floor. Sometimes alternatively, we may be able to consider what might a particular environment do to a man ***and then see whether such consequences are, in deed, to be found.***”) Osinubi Dec. at ¶1. (emphasis added); *see also* *Milward v. Acuity Specialty Prods. Group*, 639 F.3d 11, 17 (1st Cir. 2011) (“None of my nine viewpoints can bring indisputable evidence for or against the cause-and-effect hypothesis and none can be required as a sine qua non.”) (quoting Austin Bradford Hill); Federal Judicial Center, Reference Manual on Scientific Evidence 600 (3d ed. 2011) (“There is no formula or algorithm that can be used to assess whether a causal inference is appropriate based on these guidelines. One or more factors may be absent even when a true causal relationship exists.”). Dr. Osinubi, for her part, acknowledged the Bradford Hill viewpoints and provided a sound basis for her alternate methodology.<sup>7</sup>

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<sup>7</sup> Defendants’ invocation of the latinism “*post hoc, ergo propter hoc*,” *see* Def. Br. at 10, is unavailing. Dr. Osinubi does not base her opinion that Morris’s exposure to vinyl chloride was substantial solely as a result of his symptomology; she bases it on evidence that Morris was situated in an area pummeled by mass quantities of vinyl chloride gas.

**D. Vinyl chloride odor thresholds provide an acceptable basis for assessing exposure .**

Defendants' argument that "[o]dor threshold is not a valid and reliable technique for determining" chemical exposure, Defs. Br. at 12 (citing only their own expert) is false.<sup>8</sup> Courts routinely recognize that odor threshold is admissible as evidence of exposure when actual measurements are not available. *See, e.g., Taylor v. Union Pacific Railroad Co.* 2010 U.S. District LEXIS 96802, at \*12-12, 24-26 (S.D. Ill. Sept. 16, 2010) (holding that experts could conclude the exposure to sulfuric acid in excess of OSHA limits occurred because the odor threshold was at least the OSHA limit and multiple workers could smell the odor); *BP Amoco v. Flint Hills Res.*, 2009 U.S. Dist. LEXIS 131282, at \*16 (N.D. Ill. June 3, 2009) (odor threshold testimony deemed non-speculative and admissible); *Magistrini v. One Hour Martinizing Dry Cleaning*, 180 F. Supp. 2d 584, 614 (D.N.J. 2002) (same). *Roney v. Gencorp*, 2009 US Dist. LEXIS 85816 (S.D.W.V.) (denying *Daubert* challenge of expert who relied on odor threshold to estimate vinyl chloride exposure); *Lewis v. Airco*, 2011 NJ Unpublished LEXIS 191400 at \*20 (App. Div.) (recognizing odor threshold as relevant to estimate exposure to vinyl chloride).

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<sup>8</sup> It is also disingenuous insofar as at least one of the Defendants has taken the exact opposite position in prior litigation. *Cf. Sunnycal v. Csx Transp., Inc.*, 926 F. Supp. 2d 988, 998 (S.D. Ohio 2013) ("CSX argues that Dr. Green should have been allowed to testify about the odor threshold of 'chlorine gas' . . .").

While Dr. Osinubi acknowledged a potential deficiency in using odor threshold with respect to a single, unique ‘smeller,’ she *also* testified that “most people” would smell the chemical if the exposure were high enough. *See* Cuker Dec. Ex. G, Dr. Osinubi Dep. at 88-89. And the exposure levels here *were* high enough, because a sizable portion of the surveyed population reported smelling and tasting unusual odors on the day of the derailment. *See* Cedar Dec. Exhibit X, NJ DOH Health Consultation, p. 8.<sup>9</sup>

**E. Dr. Osinubi’s review of the New Jersey DOH survey does not diminish the reliability of any of her opinions.**

The DOH survey results are plainly admissible as public reports under Fed. R. Evid. 803(8), and any challenge to the methodology used by the NJ DOH goes only to the *weight* the survey evidence should be ascribed. *Cf. In re Nautilus Motor Tanker Co.*, 85 F.3d 105, 113 (3d Cir. 1996) (“[P]ublic reports are presumed admissible in the first instance and the party opposing their introduction bears the burden of coming forward with enough ‘negative factors’ to persuade a court that a report should not be admitted.”); *Ellis v. Int’l Playtex, Inc.*, 745 F.2d 292, 303 (4th

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<sup>9</sup> Defendants mischaracterize Dr. Osinubi’s testimony regarding Morris’s exposure time. *Compare* Defs. Br. at 12 (claiming Dr. Osinubi testified that from Morris’s description, “he was in the area where there was potential for high vinyl chloride exposures for around thirty (30) minutes.”) *with* Osinubi Dep. 2 at 196 (Counsel: “Mr. Morris didn’t tell you that he was in the fog for three or four minutes?” Dr. Osinubi: “No, he didn’t . . .” Counsel: “[Y]ou report that Mr. Morris estimated that it took him approximately 15 to 20 minutes to drive through one mile in Paulsboro, right?” Dr. Osinubi: “Yes.”). In any event, his emergency room record documents the 15 minute exposure. Cuker Dec. Ex. B

Cir. 1984) (“Playtex’s concern about the methodology of the studies should have been addressed to the relative weight accorded the evidence and not its admissibility.”). Defendants speculate that the survey evidence was “biased,” but that is hardly a sound basis for excluding a public survey. *Cf. Ellis*, 745 F.2d at 303 (“[A]llegations of bias are purely speculative. All epidemiological studies that might implicate a manufactured product are conducted with the possibility of litigation on the horizon.”).

Moreover, Defendants mischaracterize the evidence by saying the survey was “self administered” when, in fact, there were two surveys, and the door-to-door survey which had very similar results, was not self administered. Nor is the result from the survey “counterintuitive” because “in some instances reported symptoms increased as the distance from the derailment site increased.” As Dr. Osinubi explained in deposition, decay products are a potential cause of the symptoms complained of, but they may not form until the vinyl chloride traveled some distance from the immediate area. *See Cuker Dec. Ex. G, Osinubi 4/24/15 dep at 130-132*. In any event, the overall pattern strongly shows symptoms were much more prevalent in people living within 3,500 feet of the derailment than those who live further than 3,500 feet and certainly further than 4,500 feet. *Id.*

**F. The absence of studies or literature assessing identical high-level, non-occupational acute exposure to vinyl chloride monomer does not at all render Dr. Osinubi’s general causation opinion inadmissible.**

There is no requirement “that a medical expert must always cite published studies on general causation in order to reliably conclude that a particular object caused a particular illness.” *Heller v. Shaw Indus.*, 167 F.3d 146, 155 (3d Cir. 1999); accord *Kudabeck v. Kroger Co.*, 338 F.3d 856, 862 (8th Cir. 2003). That precedent—which by itself should permit the Court to skip Defendants’ argument at pages 17-18 of their brief—was applied in *Best v. Lowe’s Home Ctrs., Inc.*, 563 F.3d 171 (6th Cir. 2009), where the Sixth Circuit reversed exclusion of plaintiff’s medical expert, Dr. Moreno. The court stated:

Based on his medical knowledge, Dr. Moreno compiled a list of possible causes for the injury, including virus, accident, brain tumor, brain surgery, exposure to chemicals, medications, or an ideopathic (unknown) cause. Lowe’s strongest argument is that no published material confirms that inhalation of the chemical in Aqua EZ can cause anosmia. But ‘there is no requirement that a medical expert must always cite published studies on general causation in order to reliably conclude that a particular object caused a particular illness.’ Dr. Moreno did not arbitrarily ‘rule in’ Aqua EZ as a potential cause, but instead concluded from the MSDS sheet and his own knowledge of medicine and chemistry that the chemical it contains can cause damage to the nasal and sinus mucosa upon inhalation.

*Id.* at 180-181 (internal citation omitted).

Evidence of a chemical’s properties and known effects can be reliably applied to novel settings in the absence of medical literature directly on point. *See* Osinubi Dec. at ¶ 42 (“Defendants criticize my citation to studies of World Trade

Center victims, or occupational exposures because they are ‘dissimilar.’ But there is no large compendium of studies of effects of a 23,000 gallon release of vinyl chloride into a community of 6,000 people. Under these circumstances physicians look to analogous conditions.”<sup>10</sup>

Notwithstanding this, Dr. Osniubi has supplied a massive amount of medical literature to support her conclusions, which can be seen in her reports and Declaration.

**I. Dr. Osinubi reliably demonstrated that vinyl chloride exposure can be linked to respiratory disease, chronic liver disease, and vinyl chloride carcinogenesis.**

**G. Causation of Respiratory Disease**

It is undisputed that vinyl chloride is a respiratory irritant, and that its decay products, HCL and formaldehyde, are even more irritating. *See, e.g.,* Osinubi Dec. at ¶¶ 20-22. And there is no question that exposure to a high level of Vinyl Chloride or its decay products, hydrochloric acid and formaldehyde, can cause respiratory problems. *See Id.*

The New Jersey Department of Health Hazardous Substance Fact Sheet for Vinyl Chloride states “Inhaling Vinyl Chloride can irritate the nose, throat and

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<sup>10</sup> Quantitative and qualitative concerns about medical literature go the weight, not admissibility, in any event. *Cf. McCulloch v. H.B. Fuller Co.*, 61 F.3d 1038, 1042 (2d Cir. 1995) (holding that peer review and publication or general acceptance of an expert's theory goes to the weight of the testimony rather than its admissibility).

lungs,” *see Id.* at ¶ 12, and Dr. Greenberg agrees that Vinyl Chloride is a respiratory irritant. *See Id.* (Cuker Dec. Ex. H at 26:14-16).

Dr. Osinubi cites a wealth of peer reviewed literature to support her opinion that exposure to respiratory irritants such as vinyl chloride “can result in long term respiratory sequelae, such as asthma, reactive airway dysfunction syndrome (“RADS”), bronchiolitis obliterans or constrictive bronchiolitis and vocal cord dysfunction. RADS is associated with a vapor, mist, fume or gas exposure that is extremely high level and brief, and causes people to develop permanent respiratory problems. *See Osinubi Dec.* at ¶15 (citing Cuker Dec. Exhibit “H” at 197:13-19). (“Constrictive Bronchiolitis may occur where there is inhalation of gases, toxic fumes, or irritants such as nitrogen dioxide, chlorine gas, and mustard gas.”) (quoting *Small Airway Disease Related to Occupational Exposures* Gulatim, et al. and Clin. Pulm. Med. Volume 22, No.3, May 2013, Pg. 133).

A respected textbook in occupational medicine recognizes that “inhalational injury from high intensity exposures” such as those that occur in “transportation accidents” can have serious respiratory effects including irritation, inflammation, pneumonitis, asthma, pulmonary fibrosis and bronchiolitis obliterans.” *Id.* at 18-19. The same textbook confirms that “irritant induced asthma occurs ... after substantial exposure to an irritating dust, mist, vapor or fume.” *Id.* at 20. A more recent article ascribes the emerging spectrum bronchiolar disorders from

occupational and environmental exposures to a wide range of respiratory irritants.

Osinubi Dec. ¶ 30.

Hydrochloric Acid and Formaldehyde are both strongly irritating to the respiratory tract and associated with a wide range of adverse respiratory effects:

<b>Vinyl Chloride Decay Toxicants</b>	<b>Health Effects</b>
Hydrochloric acid <sup>11</sup>	<ul style="list-style-type: none"> <li>• Severe irritation of respiratory tract, characterized by coughing, choking, or shortness of breath. Severe over-exposure can result in death.</li> <li>• Inflammation of the eye is characterized by redness, watering, and itching.</li> <li>• Skin inflammation is characterized by itching, scaling, reddening, or, occasionally blistering.</li> <li>• Repeated or prolonged exposure to spray mist may produce respiratory tract irritation leading to frequent attacks of bronchial infection.</li> </ul>
Formaldehyde <sup>12</sup>	<ul style="list-style-type: none"> <li>• Corrosive: It causes skin irritation which may range from mild to severe with possible burns, brownish discoloration of the skin, urticaria, pustulovesicular eruptions, irritant and/or allergic dermatitis (eczema).</li> <li>• May be absorbed through the skin with symptoms paralleling those of ingestion.</li> <li>• Causes irritation of the respiratory tract (nose, throat, airways). Symptoms may include dry and sore mouth and throat, thirst, and sleep disturbances, difficulty breathing, shortness of breath, coughing, sneezing, wheezing rhinitis, chest tightness, pulmonary edema, bronchitis, tracheitis, laryngospasm, pneumonia, and palpitations.</li> </ul>

<sup>11</sup> Source: Hydrochloric Acid Material Safety Data Sheet.

<http://www.sciencelab.com/msds.php?msdsId=9924285>

<sup>12</sup> Source: Formaldehyde Acid Material Safety Data Sheet.

<http://www.sciencelab.com/msds.php?msdsId=9924095>

Vinyl Chloride Decay Toxicants	Health Effects
	<ul style="list-style-type: none"> <li>• Increased risk of asthma and/or allergy observed in humans breathing 0.1 to 0.5 ppm</li> <li>• Eczema and changes in lung function observed at 0.6 to 1.9 ppm</li> <li>• Central nervous system (CNS) effects include: excitement, CNS depression, somnolence, convulsions, stupor, aggression, headache, weakness, dizziness, drowsiness, and/or coma.</li> <li>• Causes gastrointestinal irritation with nausea, vomiting (possibly with blood), diarrhea, severe pain in mouth, throat and stomach.</li> <li>• Damages the kidneys, liver, central nervous system</li> <li>• Classified as carcinogenic by IARC, ACGIG, NTP.</li> </ul>

Injuries caused by vinyl chloride and its decay products are analogous to injuries caused by relatively low-dose exposures to chlorine, *e.g.* in a swimming pool which generates hydrochloric acid. *See* Osinubi Dec. at ¶ 17 (citing *Short term respiratory effects of acute exposure to chlorine due to a swimming pool accident* OEM 2001; 58 399-404) (recent reports have documented long term effects, such as asthmatic reactions, bronchial hyper responsiveness, and reduced lung function among exposed people both in general and in the work environment).

Dr. Osinubi applied this wealth of scientific literature to the following fact pattern of Mr. Morris.

"Mr. Morris ... upon intense vinyl chloride exposure, developed irritative respiratory and mucous membrane syndromes inclusive of difficulty breathing/shortness of breath, skin or eye irritation, headaches and/or dizziness." Osinubi Dec. ¶ 21.

In the case of Mr. Morris, he had immediate onset of respiratory distress following his exposures to the VC cloud from the train derailment requiring two emergency room visits on the day of the exposure (11/30/2012). Although the chest x-ray on the day of exposure was normal, the first available lung function tests showed FVC -(3.92L) 85% predicted; FEV1 - (1.82) 48% predicted; FEV1-56% ; FEF 25-75 - 27% predicted; PEF -33% predicted; with no significant bronchodilator response. ***Interpretation- lung age is 108 years. Testing indicates moderate obstruction.*** Osinubi Dec. ¶31.(emphasis original)

Following the VC exposure, Mr. Morris continued to complain of persistent dyspnea on exertion, and was not evaluated with a HRCT until 2/20/2015. This test showed small focal areas of hazy ground glass in the anteriomedial aspect of the right middle lobe and posterior aspect of the right lower lobe. ***Both of these objective diagnostic findings are consistent with what has been described in small airways/bronchiolar disease following exposures to irritant chemicals (inclusive of hydrochloric acid)*** - as per the recent article by NIOSH. (See Exhibit “O”, Cummings & Kreiss Seminars in Respiratory and Critical Care Medicine Vo; 36, No 3, 2015). *Id.* at ¶32. (emphasis added).

Dr. Osinubi has good grounds to analogize vinyl chloride to other solvents and respiratory irritants. Members of the medical science community have rightly observed that “[s]ometimes chemicals of a common type cause a generalized adverse response. For example, nearly all organic solvents from petroleum products . . . share some (but not all) symptoms in common: ‘defatting’ of the skin following dermal exposure, and central nervous system depression . . . following relatively high levels of inhalation exposure.” *In re Stand 'n Seal, supra*, 623 F. Supp. 2d at 1375 (quoting David L. Eaton, Scientific Judgment and Toxic Torts -

A Primer in Toxicology for Judges and Lawyers, 12 J.L. & Pol’y 5, 10 (2003)).

#### **H. Chronic Liver Disease**

Vinyl chloride is a known hepatotoxin and has been linked to liver cirrhosis for many years. *See* Cedar Dec. Exhibit “AA” and “AC”. As Dr. Osinubi notes in her report, the term toxicant-associated steato hepatitis (TASH) was coined to explain liver toxicity found in vinyl chloride workers “which was not explained by obesity or alcohol.” Osinubi Dec. ¶47.

Defendants cannot legitimately dispute that vinyl chloride is an independent risk factor for liver cirrhosis and that occupational studies, in addition to the *Saad* study, have shown a relationship between vinyl chloride and toxic acute exposure. IARC has not put any threshold on its conclusion that “vinyl chloride increases the risk for liver cirrhosis” and the *Mastrangelo* study forcefully concluded that vinyl chloride is “an independent risk factor” for cancer and liver cirrhosis. *See* Mastrangelo, et al., p. 1191-92 (calculating that at a level of exposure of 1,000 ppm for a time period of one year, vinyl chloride increased the risk of hepatocellular carcinoma by 1.71 and liver cirrhosis by 1.37). Cedar Dec. at AC.

#### **I. Increased Risk of Cancer Justifies Medical Monitoring**

Dr. Osinubi’s opinion that the massive short term exposure to vinyl chloride justifies medical monitoring because of increased cancer risk is supported by a substantial body of scientific evidence:

1. OSHA requires medical monitoring for workers exposed to vinyl chloride and specifically requires any employee who is exposed to a “massive release of vinyl chloride” as a result of a catastrophic mishap “shall be afforded appropriate medical surveillance.” 29 CFR §1910.1017(b)(5) and §1910.1017(k)(3).
2. The National Academy of Sciences has calculated a 1 in 10,000 cancer risk associated with the following short term exposures to vinyl chloride: 1 in 10,000 for a 30 minute exposure of 2,990 ppm or a one hour exposure of 670 ppm; or 1 in 10,000 for a 30 minute exposure of 1,180 ppm or a one hour exposure of 350 ppm. Osinubi Dec. ¶47.
3. In a study of cancer induction following single and multiple exposures to vinyl chloride, a single exposure to a high dose was found to cause cancer in mice, leading the author to conclude “one dose is sufficient if the dose is high enough.” *Cancer Induction Following Single and Multiple Exposures to a Constant Amount of Vinyl Chloride Monomer*, R. M. Hehir, *Environmental Health Perspectives* Vol. 41pgs. 63-72, 1981<sup>13</sup>.
4. Using an EPA algorithm for assessing excess cancer risk, Dr. Greenberg admitted that a 60 minute exposure at 4,800 ppm would result in an excess cancer risk of 1 in 11,170. At 90 minutes the incidence would be 50% higher or about 1 in 7,000.<sup>14</sup>

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<sup>13</sup> Dr. Osinubi’s opinion cannot be excluded simply because she relied on animal studies. *In re Paoli R.R. Yard Pcb Litig.*, 35 F.3d 717, 743 (3d Cir. Pa. 1994)

<sup>14</sup> Dr. Greenberg’s initial calculation using the EPA algorithm was one excess cancer in 333 Million, but he later admitted that this was mistaken by a factor of 1,000 and the actual increased cancer risk is one in 333,000. He based this upon an average exposure of 372 ppb, or 0.968 mg/m<sup>3</sup> over an 18 day period. Cuker Dec. Exhibit “H”, Greenberg Exhibit 12, 6/16/15 and Greenberg 6/16/15 Dep. at 105:5-109:7. Because Dr. Greenberg’s cancer risk assessment did not include any readings taken before the afternoon of November 30, *Id.* at 114:11-22, he was asked to assume that in lieu of the exposures he calculated over the 18 days, there was a single exposure of 4,800 ppm for an hour and asked to calculate an increased cancer risk on that. Using the same EPA algorithm, he concluded the increased cancer risk would be 29.81 times greater than the 1 in 333,000 cancer risk he had calculated earlier, which results in an excess cancer risk of 1 in 11,170. *Id.* at 114:23-122:25. Dr. Greenberg then admitted that if the exposure assumed was 4,800 ppm for an hour and a half, the cancer risk would go up by 50%, which

Even more concerning is the recent study of chromosomal aberration in persons exposed to a vinyl chloride train wreck in German. *Cf.* Osinubi Dec. ¶ 48. In that case, the atmospheric concentration of vinyl chloride was not measured until 15 hours after the event, at which time it was 1 – 8 ppm, certainly no higher than what was found in Paulsboro. That study, which controlled for demographic variables and smoking, showed those exposed to vinyl chloride had a statistically significantly increase in the mean frequency of aberrant cells. Dr. Osinubi has cited other articles showing “that a single exposure is effective as multiple exposures in producing chromosome damage” and that there is a significant correlation between chromosomal aberrations and the incidence of cancer. Cuker Dec. Ex. F at p. 14. Dr. Osinubi notes that mutant biomarkers have been found even in workers exposed below OSHA’s permissible exposure limit of 1 ppm. *Id.* at 14-15. Accordingly, there is a strong scientific basis to show that increased chromosomal aberrations would be expected to increase cancer risk. Cuker Dec. Ex. E at 122:6-10 (“Chromosomal aberrations have been demonstrated to be predictive of cancer development in workers who have been exposed to vinyl chloride.”)

Therefore, for all of these reasons Dr. Osinubi’s opinions on disease

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would in turn result in an excess cancer risk of about 1 in 7,062, well in excess of the 1 in 10,000 standard universally regarded by regulatory authorities an excess risk of concern. *Id.* at 123:13-18.

causation are amply supported in record and should not be excluded.

**J. Medical Tests to Evaluate GERD are Recoverable**

Contrary to what Defendants claim, Dr. Osinubi does not opine that vinyl chloride exposure “caused” gastroesophageal reflux disease (GERD), but simply that exposure to respiratory irritants, such as vinyl chloride, formaldehyde and HCL can cause or aggravate irritant induced aerodigestive dysfunction syndrome,. Osinubi Dec. at ¶41. Indeed, an increased incidence of these disorders have been found among Persons exposed to the New York disaster area in the aftermath of the World Trade Center attacks and GERD has been specifically named as a disease for which compensation may be obtained for WTC exposures. 42 USC 300 mm-22 (a)(3)(A)(xi). Vinyl chloride, hydrochloric acid, and formaldehyde were all found in the complex mix of WTC exposures. Osinubi Dec. at 43, 45.

Dr Osinubi simply recommends additional studies to evaluate Mr. Morris for GERD, which is plainly recoverable under these circumstances. Plaintiffs are entitled to recover not only for injury already inflicted, but also for expenses reasonably necessary to avert further harm. *See* Restatement 2d of Torts § 919(2) (“One who has already suffered injury by the tort of another is entitled to recover for expenditures reasonably made or harm suffered in a reasonable effort to avert further harm.”).<sup>15</sup> Even if the testing were to show that Plaintiff’s GERD had not

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<sup>15</sup> The first illustration to Section 919(2) is directly applicable here. “A negligently

been exacerbated by exposure to vinyl chloride, still the testing in order to avert further harm will have been reasonable. Thus, Plaintiff is entitled to recover for it as an element of damages, and Dr. Osinubi's opinion concerning the need to test is properly admissible. *See Grassi v. Pennsylvania R. Co.*, 86 N.J. Super. 48, 55, 205 A.2d 895, 899 (App. Div. 1964) (It was not error to permit plaintiff's medical witness to testify that plaintiff's weekly treatment should continue, to guard against further injury occurring). *See also Theobald v. Angelos*, 40 N.J. 295, 304, 191 A.2d 465, 470 (1963) ("reasonable compensation" includes all expenses reasonably necessary or incidental to plaintiff's efforts to cure or alleviate his injuries).

**III. Defendants lodge no credible challenge to Dr. Osinubi's differential diagnosis. Her opinion on specific causation is plainly admissible.**

The reliability of differential diagnosis has been approved in this circuit. *Heller, supra*, 167 F.3d at 154-155 (3rd Cir 1999); *Paoli III*, 35 F.3d 717, 742, n.8 (3rd Cir. 1994). "To properly perform a differential diagnosis, an expert must perform two steps: (1) 'Rule in' all possible causes of [injury] and (2) 'Rule out' causes through a process of elimination whereby the last remaining potential cause is deemed the most likely cause of [injury]." *Feit v. Great W. Life & Annuity Ins. Co.*, 271 Fed. App'x 246, 254 (3d Cir. 2008). Furthermore, expert testimony on

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hits and bruises B's leg. B applies a dressing to the wound but, reasonably believing that the bone in his leg may be fractured, has X-ray photographs taken. These reveal no fracture. B is entitled to recover the expense of the X-ray photographs." Restatement 2d of Torts § 919(b), cmt. b, illustration 1.

causation is not inadmissible “simply because it fails to account for some particular condition or fact which the adversary considers relevant.” *Creanga v. Jardal*, 185 N.J. 345, 360 (2005).

Defendants claim that “Dr. Osinubi fails to address plausible alternative causes in her report.” Def. Br. at 23. Shortly thereafter, however, Defendants concede that Dr. Osinubi “acknowledges . . . other possible causes of Plaintiff’s maladies.” *Id.* at 24. So Defendants move on to briefly critique the *method* by which Dr. Osinubi excludes potential other causes as nothing more than “her own say-so.” *Id.* But Defendants then recognize that Dr. Osinubi in fact rejected “alternative causes . . . based primarily on temporality.” *Id.* Defendants’ challenge to Dr. Osinubi’s differential diagnosis thus goes nowhere. *See also Heller, supra*, 167 F.3d at 154 (“Both a differential diagnosis and a temporal analysis, properly performed, would generally meet the requirements of *Daubert* and *Paoli*.”).

#### **IV. Defendants remaining arguments can be dismissed out of hand.**

Morris was indisputably exposed to high levels of a known human carcinogen as a result of the derailment, he required immediate medical attention, and deleterious health effects persist to this day. Because, as set forth, *supra*, Dr. Osinubi’s opinions on long-term risks associated with acute vinyl chloride exposure are well supported in the medical literature, her opinions—far from speculative, *cf.* Defs. Br. at 25—are admissible and can be used as the basis for

Morris's medical monitoring claim. *Cf. Theer v. Philip Carey Co.*, 133 N.J. 610, 627 (N.J. 1993) (“[P]laintiffs who have suffered increased risk of cancer when directly exposed to a defective or hazardous product like asbestos, when they have already suffered a manifest injury or condition caused by that exposure, and whose risk of cancer is attributable to the exposure.”).<sup>16</sup>

Finally, in support of Defendants' contention that Dr. Osinubi's testimony should be excluded under Fed. R. Evid. 403 they merely parrot the rule and state: “the probative value of Dr. Osinubi's opinions is clearly outweighed by [the Rule 403] concerns.” Defs. Br. at 27. The argument is thus undeserving of review by this Court. *Cf. Nagle v. Alspach*, 8 F.3d 141, 143 (3d Cir. 1993) (declining appellate review of issue mentioned just “casually in one sentence.”).

### **CONCLUSION**

For all of the reasons given above, Defendants motion to exclude the testimony of Dr. Osinubi in this case should be denied.

Respectfully submitted,

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<sup>16</sup> The appropriateness of medical monitoring relief under New Jersey law is the subject of separate motions by Defendants.